Acute pulmonary embolism masquerading as acute myocardial infarction

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Pulmonary embolism can be extremely difficult to diagnose based on clinical presentation. Many studies have demonstrated certain electrocardiographic patterns commonly seen in pulmonary embolism, but few have described changes consistent with ST segment elevation myocardial infarction. In this report, we describe a patient who presented to the emergency department with electrocardiographic findings consistent with an anteroseptal myocardial infarction and his subsequent clinical course.

Pulmonary embolism (PE) can be extremely difficult to diagnose based on clinical presentation. An electrocardiogram (ECG) is commonly used to evaluate patients with suspected pulmonary embolism. Many studies have demonstrated certain ECG patterns commonly seen in PE, but few have described changes consistent with ST segment elevation myocardial infarction (STEMI).

CASE REPORT

A 63-year-old man was brought to the emergency department in cardiac arrest. He had a history of systemic hypertension, hyperlipidemia, and coronary artery disease, with percutaneous coronary intervention and a stent to his left anterior descending coronary artery placed 3 years earlier. The patient complained of dizziness and dyspnea prior to his syncope. Emergency services found him in pulseless electrical activity and initiated successful cardiopulmonary resuscitation, and the patient was subsequently intubated. His initial blood pressure was 70/40 mm Hg, heart rate 68 beats/min, and pulse oximetry 80% on 100% oxygen via endotracheal tube.

The initial 12-lead ECG done in the emergency department showed anteroseptal ST segment elevation *(Figure 1).* Based on his clinical presentation and ECG findings, the patient was taken for an emergent cardiac catheterization. He was hemodynamically supported by norepinephrine and an intra-aortic balloon pump. His coronary angiogram showed nonobstructive epicardial coronary arteries and a patent left anterior descending artery stent.

A bedside echocardiography was performed emergently, and it revealed a dilated right ventricle and hyperdynamic left ventricle. These findings raised a suspicion of massive pulmonary embolism. Right-sided heart catheterization revealed a right ventricular pressure of 80/30 mm Hg, and a pulmonary angiogram disclosed massive filling defects suggesting bilateral pulmonary embolism *(Figure 2)*. The patient was fully anticoagulated during the procedure with intravenous heparin with an activated clotting time of >500 sec. He was considered too unstable to be sent for a computed tomographic angiogram. Based on the massive clot burden, as diagnosed by the pulmonary angiogram, surgical intervention was considered to be most appropriate.

A pulmonary embolectomy was performed, and thromboembolic material was removed. During the surgical intervention, the patient was placed on an extracorporeal membrane oxygenator for cardiopulmonary support. Unfortunately, his condition deteriorated and he died the following day. Autopsy showed multiple residual emboli *(Figure 3).*

DISCUSSION

This case highlights the fact that pulmonary embolism still remains one of the biggest masqueraders in medicine. It has an estimated annual incidence of 600,000 and is believed to cause between 50,000 and 200,000 deaths annually (1).

The ECG changes associated with PE include sinus tachycardia (the most common abnormality), complete or incomplete right bundle branch block, a right ventricular strain pattern (T wave inversions in the right precordial leads [V1–4] ± the inferior leads [II, III, aVF]), a right axis deviation, or an SI QIII TIII pattern (a deep S wave in lead I, Q wave in III, and inverted T wave in III) (2). The presence of ST elevation is rare and usually suggests massive emboli.

There are a few suggested mechanisms for the presence of ST elevation in massive PE (3–5). The sudden elevation of right ventricular pressure and consequently increased right ventricular afterload produced by pulmonary artery outflow obstruction results in right ventricular failure and dilatation

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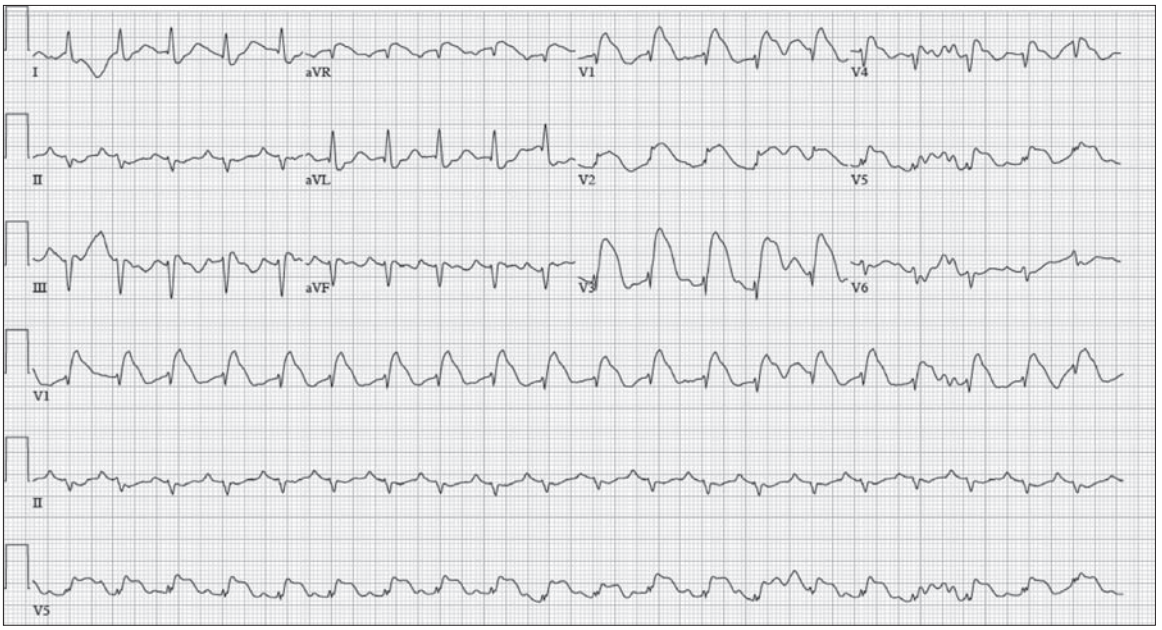

Figure 1. Presenting ECG showing ST elevations in the anterior leads.

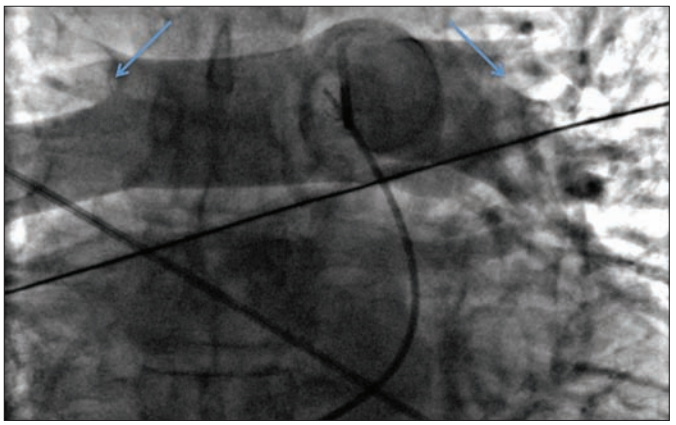

Figure 2. Pulmonary angiogram showing areas of massive bilateral filling defects suggesting pulmonary embolus (arrows).

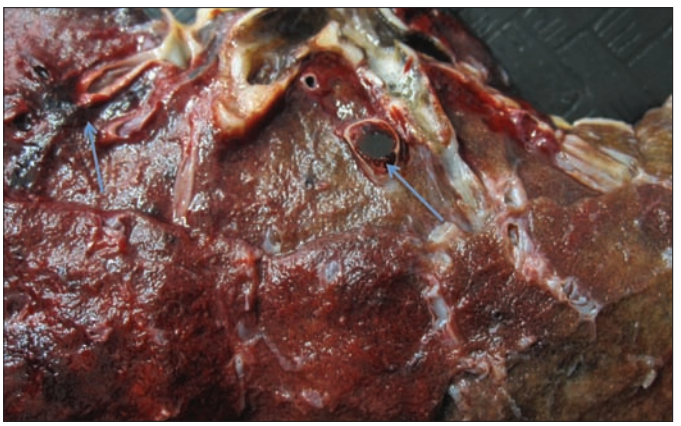

Figure 3. Gross specimen of pulmonary vasculature revealing the presence of residual thrombi (arrows).

inducing myocardial ischemia. These ST elevations could also be explained by a sudden increase in pressure on the right ventricle resulting in stretching of the myocardial cells leading to ischemia, and acute coronary vasospasm, resulting in ST elevation. The severe hypoxemia that accompanies massive PE induces a catecholamine surge and further increases myocardial workload, worsening the ischemia.

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